

A CASE OF TUMOUR OF THE PONS.

BY J. DIXON MANN, M.D., F.R.C.P.

Physician to the Salford Royal Hospital,

AND

SHERIDAN DELÉPINE, M.B., B.Sc.

Professor of Pathology in The Owens College.

WILLIAM S., aged 52 years, was admitted into the Salford Royal Hospital on August 11, 1893. He enjoyed good health until March, 1892, when he began to have shooting pains in the back of the head; the pains were subsequently felt at the top and the front of the head. He vomited occasionally. In December, 1892, he began to have humming noises in the left ear. In April, 1893, he felt a pain in the right shoulder, which extended down the arm to the hand. He has muscular twitches in the left leg and he feels rather dizzy. After remaining in hospital for several weeks, he was discharged at his own request, but he continued to attend as an out-patient. The right arm and right leg now began to feel weak, and in September, 1894, he had ptosis of the left eye, which lasted three months. In the early part of December he had a succession of convulsive attacks. The noise in the left ear has been continuous.

On February 4, 1895, he was re-admitted, when his condition was as follows:—The right arm, flexed at a right angle, was spastically contracted and devoid of motor power. The right leg was also spastic and devoid of motor power. There was no paralysis of the facial muscles and the ptosis had disappeared—the left eye could be freely opened. There was no perceptible paralysis of the ocular muscles—the eye ball was capable of the usual movements, nor was there any nystagmus. The pupils were equal, not contracted, and they reacted to light. There was double optic neuritis and slight limitation of the field of

vision on the temporal side of the left eye; vision was otherwise unaffected. The humming or buzzing sound in the left ear was now associated with impaired audition on the same side. The perception of acid substances applied to the tongue was equally good on both sides. The pain in the head had almost disappeared. When tested with the point of a pin and with test-tubes respectively filled with hot and cold water, sensation was found to be unaffected on both sides of the face, on the arms and on the legs. On the right side the knee-jerk was slightly increased. The speech was much impaired, being thick and "bulbar"; there was also some difficulty in swallowing. The mental faculties were unaffected.

Whilst in hospital the patient had twenty to thirty seizures, in which, for the most part, the head and the eyes were turned to the right side, the left eye being less deflected than the right. When the patient was in a passive condition, no difference was perceptible between the movements of the left eye and those of the right. The seizures were characterised by a condition of tonic spasm, which lasted from a few seconds up to two or three minutes, the right arm and the right leg being absolutely stiff and rigid. The left arm and leg were not affected. The pupils were dilated and did not respond to light. The respirations were slowed. During the seizures and for a varying period afterwards—from a few minutes to half an hour—the patient was quite unconscious. On one or two occasions there were slight indications of an epileptiform character, and once, slow clonic spasms—one every four or five seconds—were observed to affect the right arm. In two of the seizures the right side of the face appeared to be partially paralysed, the cheek "flapping" with the respirations at the maximum of the attack. In February, 1895, both sides of the face, both arms, and both legs twitched during an attack. On another occasion the twitching was limited to the fingers of the right hand, the face not being affected. On March 21 the left eye was observed to remain closed for about twelve hours after an attack. During the interval between the attacks, if the patient (who was then quite conscious and intelligent) was asked a question, the answer to which involved some reflection, the left eyelids usually twitched convulsively. After a seizure on March 27, ptosis on the left side recurred and persisted until death, which took place on April 4, 1895. No sugar was found in the urine, nor albumin until a fortnight before death.

*Professor Delépine's Report on the brain, cerebellum, pons
and medulla oblongata.*

These parts having been kept in weak spirit for a few days before being available for examination were very soft, and had partly lost their original colour. The only evidences of disease which could be detected by an external examination were the following:—

(1) A marked swelling of the left side of the pons, most distinct above the exit of the 5th nerve, and causing much asymmetry of the pons.

(2) A very considerable enlargement of the left peduncle of the brain.

(3) Great displacement of the basilar, left posterior cerebral and left superior cerebellar arteries as well as of the left 3rd and 4th nerves, and to a lesser degree of the 5th nerve.

(4) Atrophy of the left 3rd nerve, which measured about 2 mm. in diameter, whilst the right 3rd measured over 3 mm. in diameter.

(5) On separating slightly the left temporo sphenoidal lobe from the left crus, a tumour became apparent. Owing to the soft state of the lateral portion of the left crus, and of consequent lacerations of this part, the superficial relations of this tumour could not be very clearly ascertained. It was, however, evident that the tumour, which seemed to be of the size of a pigeon's egg, must have compressed the posterior parts of the left optic tract.

(6) When the upper surface of the cerebellum was exposed, a distinct bulging was evident in the anterior part of the left quadrate lobe. This swelling was situated about $\frac{1}{2}$ inch to the left of the superior vermiform process, which was displaced towards the right, so that, seen from above, the two halves of the cerebellum seemed unequal. The left cerebellar hemisphere looked larger than the right.

Owing to the soft state of the specimen, it was not found possible to make many sections through the parts affected. The following were, however, found sufficient to determine with tolerable accuracy the localisation of the

DIAGRAM I.

Diagram of left aspect of medulla, pons, crus cerebri, thalamus opticus, etc., to show the relations of the tumour and surrounding areas of degeneration as well as the direction of the various sections described in the text.

I to 6.—Planes of sections.

II. to VIII.—Cranial nerves.

A—Pulvinar.

B—External and internal geniculate bodies.

C—Pineal gland.

D—Corpora quadrigemina.

E—Peduncle of cerebrum.

F—Left corpus albicans.

G—Superior peduncle of cerebellum.

H—Middle " " "

K—Inferior " " "

L—Medullary centre of left hemisphere of cerebellum. The letter is placed at the confluence of the primary medullary lamellæ of the anterior part of the antero-superior lobule and of the lateral part of the central lobule.

M—Olive.

The tumour is indicated in black, and the degeneration by tight shading.

DIAGRAM II.

Section 1 (seen from behind).

A—Internal capsule.

B—Substantia nigra.

C—Optic thalamus.

C—Left optic thalamus at the place where degeneration has extended from the tegmental portion of the peduncle (degenerated part indicated by the darker shade).*

D—Lenticular nucleus.

E—Vclum interpositum.

F—Fornix.

G—Corpus callosum.

This sketch should be reversed to make it comparable with the other drawings.

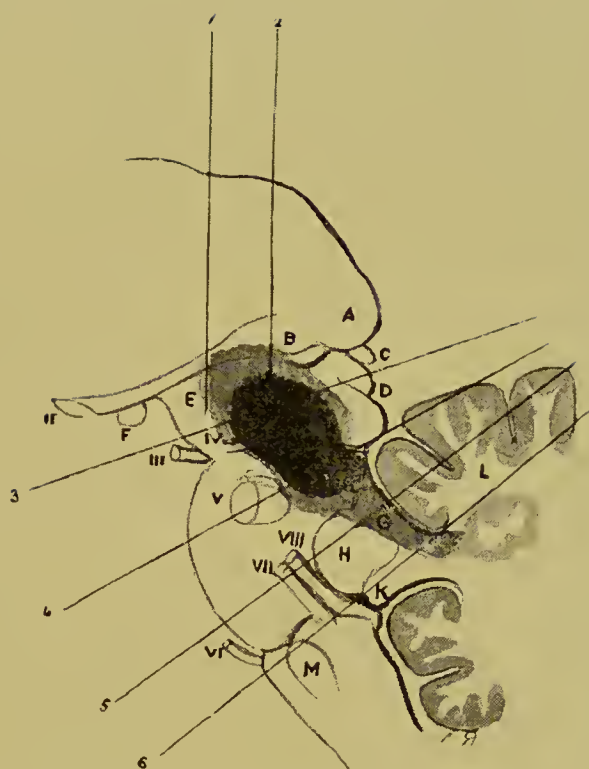


DIAGRAM I.



DIAGRAM II.

lesions, except in the left external part of the left crus, which had been destroyed by softening and *post-mortem* laceration.

The planes of section will be best understood by reference to the diagram I., which represents the posterior parts of the basal ganglia, crura cerebri, pons varolii, medulla, cerebellum, and peduncle of the cerebellum, seen from the left side. In this diagram, the affected parts are shaded, the tumour itself deeply, the softened parts less deeply. All these parts could not be seen from the surface, as has already been explained.

Section 1 (Diagram II.).—A vertical frontal section passing through the hemispheres of the brain and dividing the basal ganglia behind the grey commissure. It shows the relations of the tegmental and crustal portions of the cerebral peduncles to the region of the optic thalami and the internal capsula. A small portion of the locus niger indicates the line of demarcation between the tegmentum and crusta. A patch of degeneration is seen to have destroyed a small portion of the left optic thalamus and subthalamic region. The internal capsule is apparently unaffected.

Section 3 (Diagram III.).—This section was slightly oblique, dividing the crura just above the superior corpora quadrigemina, and the point of exit of the 3rd nerves.

A very vascular tumour, surrounded by a layer of very soft, degenerated, nervous tissue, occupies the space between the grey matter of the corpora quadrigemina posteriorly, and the crusta anteriorly, the mesian plane on the right, and the surface of the peduncle on the left.

It is at this level that part of the tumour and surrounding tissues had been accidentally destroyed, but the shape and relations of the tumour indicate that it had probably extended from the surface of the crus, and originated in the meningeal covering. There can be little doubt either that the left corpora geniculata were involved. The left crusta, except in its most external part (temporo-occipital cerebellar fibres and pyramidal fibres for the leg), was not involved, but must have been subjected at times to considerable pressure. The grey matter round the aqueduct had been simply dis-

DIAGRAM III.

Section 3.—Passing through the superior corpora quadrigemina, the aqueduct, the crusta, a little above point of exit of the third nerves (seen from above).

- A*—Superior corpora quadrigemina.
- B*—Brachium of left testis.
- C*—Crusta; letter placed opposite pyramidal fibres.
- D*—Substantia nigra.
- E*—Aqueduct.
- F*—Right third nerve.
- F'*—Left third nerve.

The relative size of the two nerves is represented by means of transverse sections, below the main diagram.

- G*—Basilar artery with its branches.
- H*—Tumour (dark shade).
- I*—Zone of softening round the tumour (lighter shade).

DIAGRAM IV.

Section 4.—Passing through pons Varolii at level of exit of fifth nerve, upper part of superior cerebellar peduncles just below the inferior corpora quadrigemina, inferior corpora quadrigemina, and most anterior and superior cerebellar laminæ (seen from above).

- A*—Superior cerebellar peduncle.
- B*—Fifth nerve.
- C*—Pyramidal bundles.
- D*—Superficial transverse fibres of pons.
- E*—Deep transverse fibres of pons.
- F*—Fillet.
- G*—Aqueduct near upper end of fourth ventricle.
- H*—Anterior end of superior vermiform process.

Tumour and degenerated parts indicated as in previous diagrams.



placed towards the right. It seems impossible that the fibres of the left 3rd nerve should have escaped destruction in their passage from their nucleus to the surface, and as a matter of fact the left 3rd was in a state of advanced atrophy.

Section 4 (Diagram IV.).—This section is slightly more oblique than the last, and followed the fibres of the 5th nerve for some distance through the pons.

In this section the lower part of the tumour is seen to extend beneath the fibres of the superior cerebellar peduncle, which seem to be partly destroyed by it. The zone of softening round the tumour extends more in the direction of the fibres of the 5th than in any other direction. The aqueduct is pushed towards the right, and part of the grey matter round it on the left side remains apparently unaffected. The right half of the tegmental and ventral parts of the pons are but slightly affected by pressure at this level, but the left half is still considerably enlarged.

Section 5 (Diagram V.).—This section is a little more oblique than the previous one. It is entirely below the level of the tumour, and shows only a tract of degenerated tissue in the region corresponding to the superior cerebellar peduncle and adjacent white matter. Though the left half of the pons is distinctly larger than the right, it seems almost entirely free from lesion.

Section 6 (Diagram VI.) is nearly parallel to the last. It passes through the upper part of the medulla oblongata, showing the exit of the 8th nerve; it also divides the floor of the 4th ventricle at about the level of the striæ acusticæ, it exposes the white centres of the cerebellum, with a small part of the corpora dentata; the relations of the central lobule are also well shown.

The only lesion visible at this level is a small patch of softening evidently following the direction of the fibres of the superior cerebellar peduncle and involving the region of the left corpus dentatum. There is a slight displacement of the central lobule towards the right; the floor of the 4th ventricle may have been at times compressed by the central lobule when the diseased parts underwent rapid enlargement from one cause or another.

DIAGRAM V.

Section 5.—Passing through the pons Varolii at a level half way between lower edge of the pons and exit of the fifth nerves (seen from above).

A—Superior medullary velum, behind fourth ventricle.

B—Middle peduncle of cerebellum.

C—Seventh and eighth nerves.

D—Pyramidal fibres.

E—Flocculus.

F—Superior or quadrate lobe.

G—Vermiform process.

Softened part shaded as in previous diagrams.

DIAGRAM VI.

Section 6.—Passing through medulla immediately below lower border of pons, through the central lobule and postero-superior lobule of cerebellum (seen from above).

A—Anterior pyramid.

B—Root of eighth nerve.

C—Central lobule.

D—Upper part of right corpus dentatum.

The patch of softening is indicated as in other diagrams.



DIAGRAM V.



DIAGRAM VI.

From a study of these sections one may safely come to the following conclusions :—

The left crus was the seat of a tumour which had destroyed the greater part of its tegmentum. This tumour had apparently originated near the external left aspect of the crus, probably extending from the membranes and inserting itself like a wedge between the left corpora quadrigemina and the crustal portion of the left cerebral peduncle, causing them to become widely separated and disconnected. This tumour did not extend upwards higher than the upper parts of the upper corpora quadrigemina, and downwards below the upper border of the middle cerebellar peduncle; the formatio reticularis and the red nucleus were the parts primarily invaded and destroyed by the tumour (within the region affected); other parts were also destroyed, *more or less* completely, being involved in the zone of degenerated and necrosed tissues surrounding the tumour; these were the left superior cerebellar peduncle, the left upper and lower fillet, the left corpora geniculata and posterior part of the left optic tract, the fibres of the root of the left third nerve; the left 4th nerve was possibly involved, in its course round the diseased crus. Many of the fibres of the left 5th must also have been involved.

The nuclei of the 3rd, 4th, 5th, were all in close contact with the tumour, and partly involved in the necrosed zone.

The fibres of the 2nd, 3rd, 4th, 5th, 6th, 7th, 8th nerves had either to pass through degenerated zones or were subjected to pressure, which must have been very variable in degree, considering the very vascular nature of the tumour. The external $\frac{1}{4}$ th of the left crusta was partly infiltrated by the tumour, and had certainly much suffered from pressure; the rest of the left crusta was also subjected to pressure, though in a lesser degree. The pressure must also have at times affected indirectly the right half of the pons and the right crus.

The left 3rd nerve was the only nerve which showed striking evidences of the destructive action of the tumour.

This nerve was much atrophied; its diameter was only $\frac{2}{3}$ ds of that of the corresponding nerve on the right side.

On microscopical examination not more than $\frac{1}{4}$ th of its fibres could be recognised, and most of these were in a state of degeneration. Connective tissue occupied the space originally occupied by the other fibres. The right third was also partly degenerated, not more than $\frac{2}{3}$ rds or $\frac{3}{4}$ ths of its fibres presenting a normal appearance.

The tumour, by its situation, its relations to the formatio reticularis, the apparent hypertrophy of the pons produced by it, might have been supposed to be a typical glioma; but it was quite clearly a round-celled sarcoma. It was well defined, surrounded by a well marked zone of necrosed tissue, so soft that it was difficult to prevent a displacement of the parts. The tumour was nodulated, the nodules being firmly connected together by the condensed tissue intervening between them. The nodules were soft, very vascular, and owing to their dark colour, contrasting sharply with the surrounding opaque, yellowish white, surrounding necrosed nerve tissue. There was enough of this softened tissue to reconstitute the parts of the crus which seemed to have been replaced by the tumour, so that it is very doubtful whether there was any organic connection between the new growth and any part of the tegmentum. The cells of the tumour were small, rounded, with large nuclei, not separated by any distinct stroma except quite at the periphery of the nodules, where, through pressure, the cells were arranged in rows or packets separated by a very small amount of a clear, slightly fibrillated matrix.

The vessels were large, most of them with indistinct walls composed of embryonic looking cells; in some places the vessels were surrounded by several concentric layers of these cells. Though the tumour was almost angiomatous in a few places, there were but few traces of hæmorrhage; there was little evidence of degeneration in the central parts of the nodules. In all these respects the tumour resembled closely similar sarcomata growing from the soft membranes of the brain.

I therefore come to the conclusion that the case is one of round-celled sarcoma (very vascular) growing from the pia mater and penetrating into the left peduncle of the

brain, causing destruction of the tegmentum at that level, pressure on surrounding parts, and degeneration of certain fibres.

On reading Professor Delépine's description it is a matter for surprise that so many of the cranial nerves escaped suffering from the effects of the lesion; for not only were most of them directly involved by the tumour, but many had also to pass through a zone of degeneration and were subjected to varying pressure due to great vascular irregularity of the degenerated area. The third was the only nerve which showed definite atrophy, the paralytic symptoms being very partial; ptosis occurred, but no external strabismus, nor any alteration in the size or mobility of the pupil, nor in the power of accommodation. Lesions of the third nerve, however, often produce only partial results. No facial pain nor anæsthesia occurred from first to last, although the fifth nerve and its nucleus came well within the influence of the tumour; the seventh nerve also escaped, notwithstanding its dangerous contiguity. The slight indications of paralysis of the right side of the face on two occasions were due to diffuse pressure from increased vascularity. On the other hand, subjective auditory sensations were among the first and the most persistent of the symptoms; this, in a degree, is common, as are also spasm and convulsions in chronic lesions of the pons, the very gradual compression of the fibres of the pyramidal tract rarely causing sufficient irritation as to evoke spasm. The extremely slow growth of the tumour—three years and one month from the earliest symptom—probably accounts for the extraordinary tolerance displayed to its disturbing influence by the cranial nerves.

CONTENTS.

	PAGE
 ORIGINAL ARTICLES AND CLINICAL CASES:—	
PATHOLOGY OF A CASE OF FRIEDREICH'S DISEASE. BY H. MACKAY, M.D.	435
A CASE OF TUMOUR OF THE PONS. BY J. DIXON MANN, M.D., F.R.C.P., AND SHERIDAN DELÉPINE, M.B., B.Sc.	475
A FORM OF DISEASE RESEMBLING THE PSEUDO-SCLEROSIS OF WESTPHAL AND STRÜMPPELL. BY WILLIAM G. SPILLER, M.D.	486
ON PARALYSIS AND MUSCULAR ATROPHY IN TABES DORSALIS, WITH OBSERVATIONS ON THE RELATION OF AFFERENT IMPULSES TO MOVEMENT AND MUSCULAR NUTRITION. BY ARTHUR J. WHITING, M.D., M.R.C.P.	494
ON TWO CASES OF EARLY SYPHILITIC PARAPLEGIA. BY F. PARKES WEBER, M.D., F.R.C.P.	520
AN EXPERIMENTAL STUDY OF VISIONS. BY MORTON PRINCE, M.D.	528
A CONTRIBUTION TOWARDS THE DETERMINATION OF THE ENERGY DEVELOPED BY A NERVE CENTRE. BY VICTOR HORSLEY, F.R.S., F.R.C.S.	547
CASE OF EPILEPSY WITH TASTING MOVEMENTS AND "DREAMY STATE"—VERY SMALL PATCH OF SOFTENING IN THE LEFT UNCINATE GYRUS. BY J. HUGHLINGS JACKSON, M.D., F.R.C.P., LL.D., F.R.S., AND WALTER S. COLMAN, M.D., F.R.C.P.	580
 REVIEWS:—	
DR. OTTO SCHWARZ. DIE BEDEUTUNG DER AUGENSTÖRUNGEN FÜR DIE DIAGNOSE DER HIRN- UND RÜCKENMARKS-KRANKHEITEN. (H. WORK DODD, F.R.C.S., &c.)	591
CHARLES K. MILLS. THE NERVOUS SYSTEM AND ITS DISEASES. (W. ALDREN TURNER)	592
PROCEEDINGS OF THE NEUROLOGICAL SOCIETY OF LONDON	594
 TITLE AND INDEX.	